ATRIAL DISSOCIATION

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Schrumpf (1920) was the first to describe a clinical electrocardiogram assumed to represent atrial dissociation. He figured a curve apparently normal with a series of rhythmic and independent deflections resembling auricular waves superadded to it. In his case these independent deflections were ineffective to evoke ventricular responses, their rate was not rapid, and they fell in varying time-relation with the waves of the fundamental rhythm which they failed to affect.

Since then a number of electrocardiograms showing essentially the same events have been published by Bay and Adams (1932), Géraudel (1935), Duclos (1935), Lian and Golblin (1938), Dominguez and Bizzozero (1937), Giraud *et al.* (1943), and Deitz *et al.* (1957).

The superadded summits have been labelled P', P'', P^2, and p, in order to denote their auricular nature and to separate them from the apparently normal conducted atrial waves which are seen in the same records. Such cases have been related with another type of pararrhythmia, assumed to depend upon the same mechanism of atrial dissociation, in which two independent, grossly abnormal auricular rhythms (auricular fibrillation plus auricular flutter), or a distinctly anomalous activity with a simple auricular rhythm co-exist.

Most authors have assumed that a completely independent activity of each atrium afforded a plausible explanation of these electrocardiograms, or, in other words, that an interauricular dissociation would be present in each example of this condition.

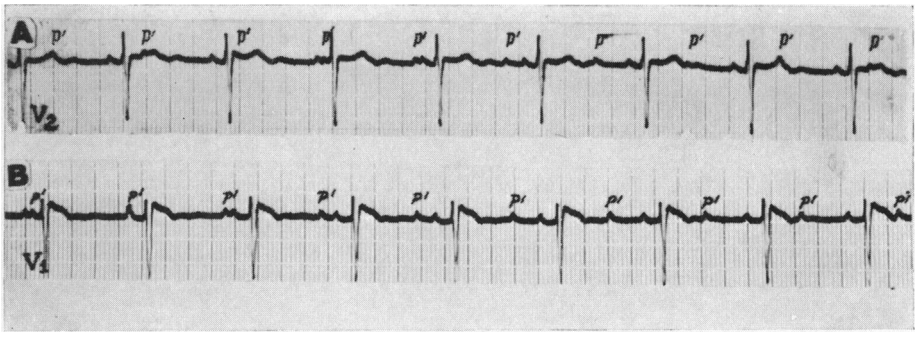

FIG. 1.—Two cases in which a series of rhythmic and independent deflections, probably auricular in origin, is seen superadded to the apparently normal dominant rhythms. Record *A* is lead V2 obtained in Case 1: *B* is lead V1 obtained in Case 2. These curves illustrate the complete independence between the atrial waves conducted to the ventricles and those of the superadded rhythm. (Standardization: 1 cm.=1 mV; time interval: 0·2 sec. between heavy vertical lines.)

These conventional views harmonize with evidence presented by Condorelli (1929) and others that examples of interauricular dissociation occur in animal experiments. However, experimental precedent does not imply that electrocardiograms showing two unrelated series of waves, apparently auricular in origin, always represent a state of complete independence between the atria.

The purposes of this paper are to report two cases of this rare electrocardiographic abnormality and to discuss the concept of atrial dissociation.

Case Reports

Case 1. A woman of 76, was first seen with ten months' history of breathlessness and moderate swelling of the lower limbs. At that time she was digitalized with lanatoside C and was asymptomatic.

On examination she had very slight œdema of the ankles. There was no dyspnœa nor crepitations at the lung bases, and the liver was not enlarged. The heart sounds were normal, no murmurs were heard, the pulse was regular, and the blood pressure was 180/80 mm. Hg. Screening revealed a slightly enlarged left ventricle. At that time a cardiogram (Fig. 2) showed regular rhythm, apparently sinus in origin, at a rate of 71, in leads I and II. This rhythm was replaced in the other leads by another rhythm, probably a supranodal or "upper" nodal rhythm (rate 65). In leads V1, V3, and V6 a secondary set of atrial waves (rate 86), independent of the dominant rhythm and not conducted to the ventricles, was present.

Seven days later another electrocardiogram (Fig. 3) showed regular rhythm, probably sinus in origin (rate 68) in all leads, and a secondary set of atrial waves (rate 77) in leads V1, V2, and V3. Several subsequent records have failed to reveal the distinctive pattern of atrial dissociation or abnormal pacemakers.

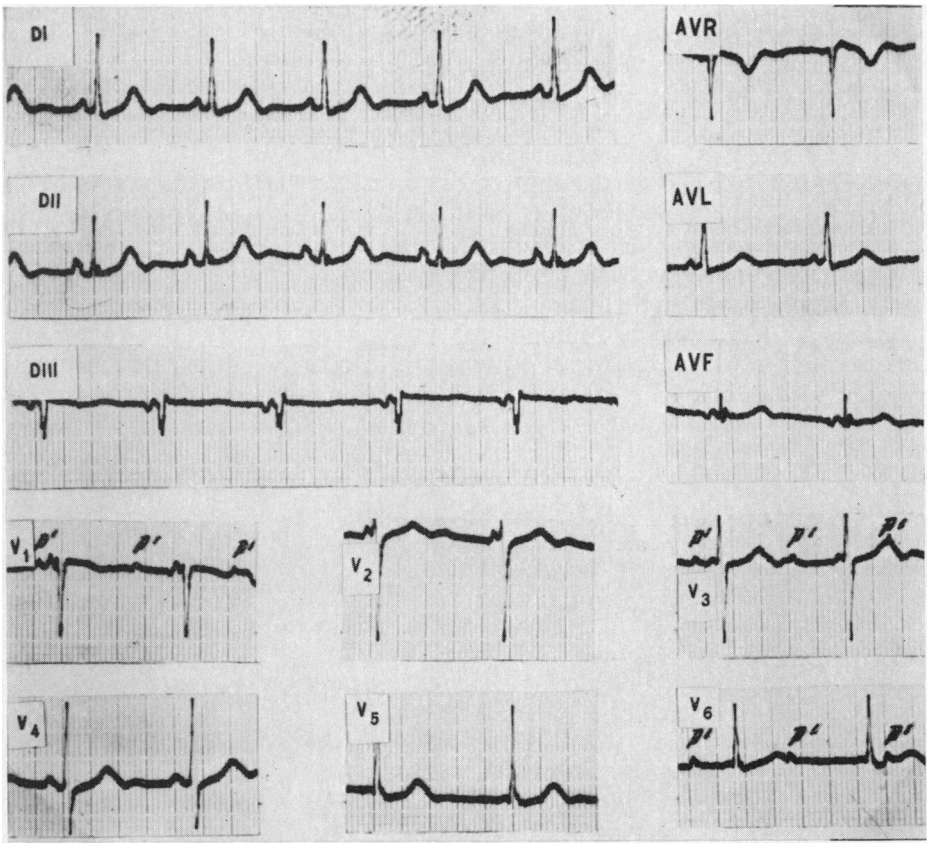

FIG. 2.—First electrocardiogram obtained in Case 1. The apparently sinus rhythm recorded in leads I and II changed in lead III and unipolar leads to a probably supranodal or upper nodal rhythm. In leads V1, V3, and V6 a secondary set of atrial waves not conducted to the ventricles and completely independent from those of the fundamental rhythm is seen.

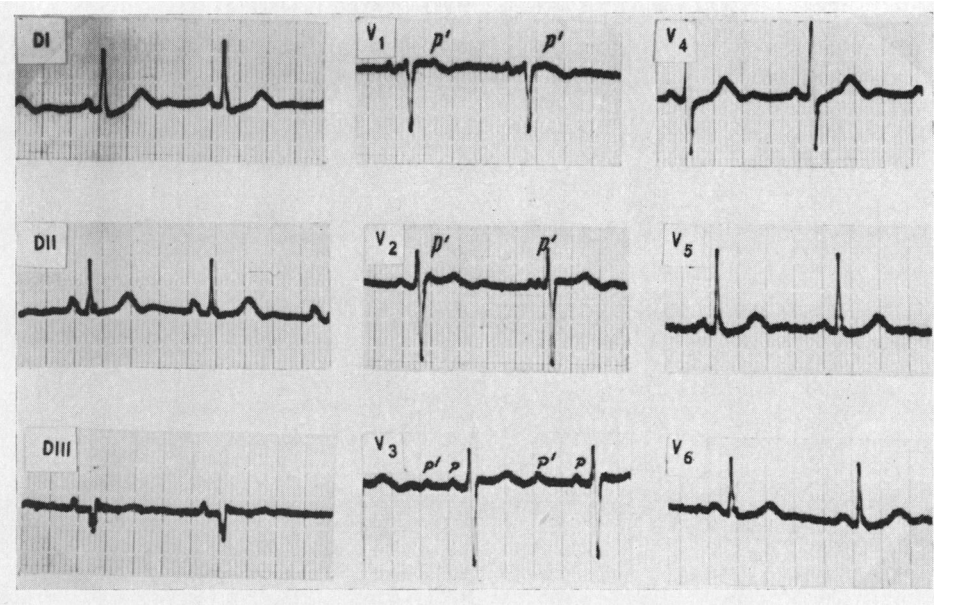

FIG. 3.—Second electrocardiogram obtained in Case 1. The fundamental rhythm is apparently sinus in origin in all leads. The superadded atrial waves are evident in leads V1, V2, and V3.

Case 2. A man of 57, was admitted complaining of severe dyspnœa and œdema. On first examination he was orthopnœic and cyanosed. The pulse was regular and the blood pressure 130/80 mm. There were a raised jugular venous pressure, crepitations at the lung bases, and evidence of severe bronchitis and emphysema. The heart sounds were normal and there were no murmurs. The liver was enlarged and there were massive œdema of the lower limbs and sacral œdema.

Roentgen examination of the chest revealed a slightly enlarged left ventricle, a convex cardiovascular angle at the left border and a calcified aortic arch. An electrocardiogram showed a regular sinus rhythm and minor evidence of left ventricular strain.

Treatment consisting of oxygen, digitalization with lanatoside C, acetazolamide and mercurial diuretics, antibiotics, and a low-salt diet resulted in prompt improvement. Relief of the dyspnœa, disappearance of the crepitations at the bases of the lungs and of œdema were obtained in one week. At that time a second record (Fig. 4—I) showed a regular rhythm, apparently sinus in origin, at a rate of 64. A secondary set of atrial waves, independent of the dominant rhythm and not conducted to the ventricles, was present in leads V1, V2, V3, and V5 (rates of 54 to 66). A third record (Fig. 4—II) eight hours later showed the same secondary activity in leads V1, V2, and V6 (rates of 56 to 83). At that time an œsophageal electrocardiogram at various levels failed to reveal the superadded atrial activity, a point that suggests it was right atrial in origin, since immediately before and after it was portrayed in præcordial leads, particularly in those recorded from the right chest. Twenty-four hours later the super added waves were present (Fig. 4—III) in leads V1, V5, and V6 (rates of 71 to 77).

Several subsequent records, including an intracardiac electrocardiogram at various right atrial levels failed to reveal the existence of atrial dissociation.

DISCUSSION

Close inspection of many published electrocardiograms claimed to demonstrate two simultaneous and independent atrial activities does not permit acceptance of the evidence as conclusive. Thus, extraneous somatic waves in cases with sinus rhythm, technical artifacts, and other sources of error or a misconception of the nature of the large auricular waves in right chest leads in patients exhibiting typical fibrillation waves in left chest and limb leads, are frequently misinterpreted in early papers on the subject and described as electrocardiographic evidence of atrial dissociation.

However a few published electrocardiograms are convincing like those showing two independent sets of atrial waves, the tracings published by Lombardini and Aviles (1939) and Bellet (1953) as

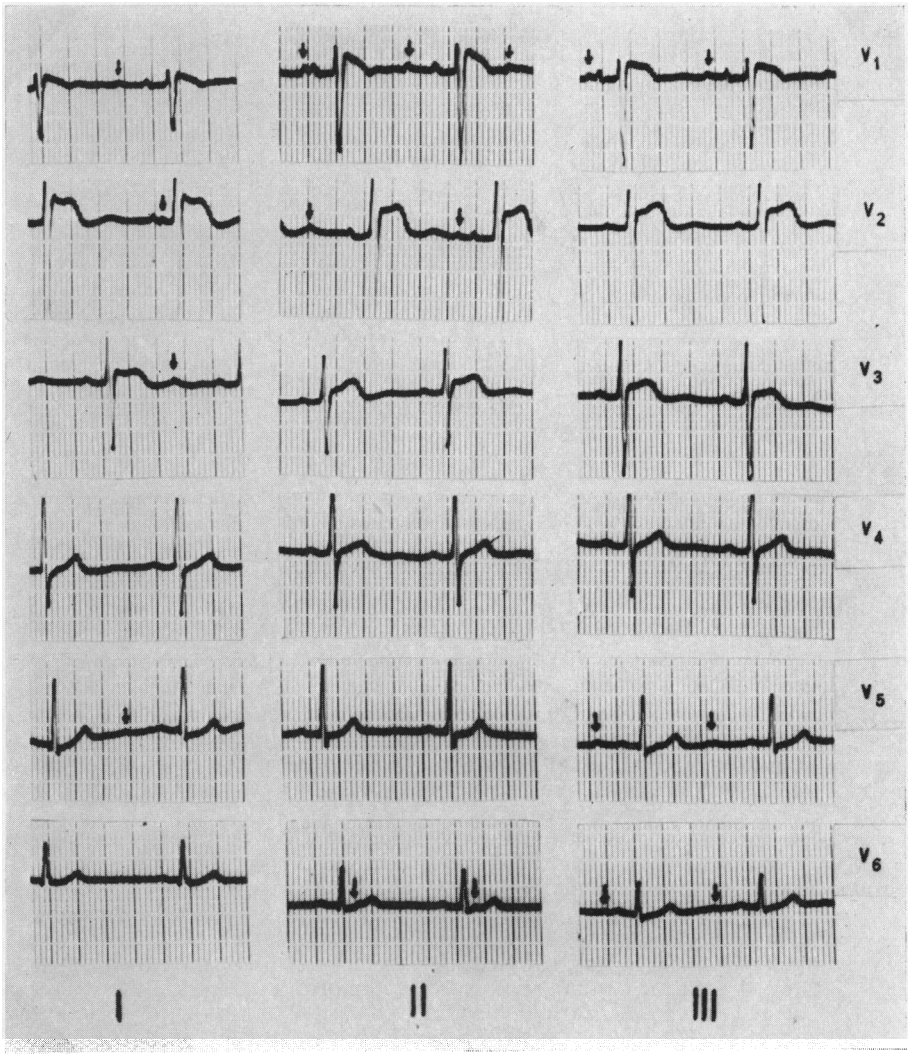

FIG. 4.—Unipolar chest leads taken at different times in Case 2. The three records demonstrate the variability of the rate of the superadded rhythm from lead to lead in the same record.

examples of simple atrial activity with superimposed atrial flutter, the tracing published by Mussafia and Jacovella (1957) as a case of simple activity (probably sinus rhythm) with simultaneous paroxysmal atrial flutter and those published by Araújo Moreira (1951) where the fundamental mechanism is that of auricular fibrillation while a simple, not rapid, atrial rhythm is evident in præcordial leads.

Two further cases were recently described by Deitz *et al.* (1957). They are remarkable, since they provide an illustration of transitional stages between simple dissociation, where two independent series of slow atrial waves co-exist, and more complex pararrhythmias in which the superadded rhythm is auricular flutter or auricular fibrillation.

In all these cases the cardiac nature of the superadded electrical activity seems probable and its atrial origin is supported by the contour of the oscillations. These are best recorded by placing the exploring electrode over the right chest, sternum, or at various œsophageal levels. This seems to be another point that suggest an atrial origin for the superadded waves.

A review of the associated disturbances of rhythm in cases with atrial dissociation indicates that

a variety of arrhythmias is observed in this curious electrocardiographic phenomenon. Ectopic premature systoles are seen to occur as in any other condition, and may be nodal or ventricular in origin. Supranodal or "upper" nodal rhythm and second degree A-V block are also seen to occur. In general the dominant rhythm has the usual average rate for each particular case. In those cases showing two independent simple rhythms the auricular rates of the dominant rhythm are sometimes faster and sometimes slower than that of the auricular superadded deflections.

Superimposed auricular activities are better displayed in different leads in different patients, the contour of the atrial waves varies from patient to patient, and in several cases these waves are not portrayed in the limb leads. In our cases the leads where the specific pattern of atrial dissociation is identified change from one record to the next. This pre-supposes, if a true interauricular dissociation is assumed, a change of direction of the spread of the excitation. It may be said that the disparity in the electrocardiographic findings in atrial dissociation is caused by the position of the heart in the chest, by the site of the ectopic pacemakers, and by the manner in which the excitation waves are distributed to the auricular syncytium. This does not explain why and how the ectopic impulse could spread over the whole surface of an atrium without being recorded in the limb leads, even when the spread of the excitation wave changes its direction from time to time.

From these considerations it seems unwise to accept the interauricular dissociation as an adequate explanation for all cases under discussion. It is of interest to remember that Lewis (1925) discarded the hypothesis of an interauricular dissociation as an explanation for the Schrumpf's case. However, a few years later Condorelli (1929) found that he could induce an interauricular dissociation in dogs by ligating the interatrial branch of the left coronary vessel. By this method he found that dissociation of the two atria develops with sinus rhythm in the right atrium and fibrillation in the left atrium. Condorelli also found an interatrial dissociation with sinus rhythm in the right atrium and left atrial arrest by ligating in a dog the anterior descending branch of the left coronary artery proximal to the origin of the interauricular branch which sometimes arises from that vessel. Dissociation of the two atria has been supported by the investigations of Scherf and Siedek (1934). A unique convincing direct observation of this phenomenon in human beings has been reported by Marcel and Exchaquet (1938). Their studies on the electrocardiographic patterns of human fœtus without possibility of survival were carried out with open chest, and, this way, they were able to correlate the electrical and mechanical events of the cardiac cycle. Analysis of their electrocardiograms strongly suggests that atrial dissociation occurs in human beings. However, the applicability to clinical conditions of results obtained in such work is doubtful and it seems, at the present stage, that dissociation of the two auricles in human beings cannot be considered as established. None of the observations made in connexion with human cases of double atrial activity includes any other evidence beside the electrocardiographic tracings. On the other hand, it is not possible to find any observations that could not be understood as the result of the spread of impulses over circumscribed areas of the atria.

In view of such reasons the use of the term "interauricular dissociation" for this kind of pararrhythmia does not seem justified and it would seem better, from a clinical point of view, to use the wider terms "atrial dissociation" or "intraauricular dissociation." The observations of Lewis (1925), Prinzmetal *et al.* (1952), Puech (1954), and Abildskov *et al.* (1955) led them to conclude that the excitation wave spreads through the atria in a uniform radial fashion.

If a zone of refractory tissue has to be invoked in cases of atrial dissociation, those who accept an independent activity of each atrium as an explanation to these cases have to conceive a block zone running along the line of junction of the two atria. This, however, seems highly improbable. It is possible that different mechanisms are responsible for atrial dissociation, while occasionally it may be due to an interauricular block. A plausible explanation of many cases could be afforded by assuming that they are due to the simultaneous activities of two or more centres producing impulses that yield independent excitation processes of different parts of the atrial myocardium, with a dynamic bidirectional block along their boundaries.

For the purposes of this paper the term "dynamic bidirectional block" would be understood

as meaning a block guarding each centre against interference by the other centres, protecting these against the impulses of the first, and, moreover, at times changing its spatial localization in the atrial myocardium. This hypothesis presupposes that the area responding to each centre varies from time to time and, theoretically, it could be at different times, greater or less than a whole atrium. A similar mechanism was postulated by Moreira (1951) as an explanation of a repetitive paroxysmal tachycadia of certain cases.

These views have received support from experimental work eliciting dissociation of small areas of atrial muscle and particularly from the extensive clinical studies on intracardiac potentials of Giraud *et al.* (1955). These authors found, in cases with auricular fibrillation, several independent areas where the activity is rhythmic and coordinated, surrounding rhythmic foci of impulse formation. These areas are separated by wandering boundaries from the remainder of the atria, where the disorganized activity, typical of fibrillation, is present. Giraud *et al.* thus showed that two, or more, pararrhythmic centres mutually protected against each other's impulses by dynamic bidirectional blocks may be present in human atria.

It is concluded that electrocardiograms showing two simultaneous and independent atrial activities do not necessarily mean the existence of an interatrial dissociation and, commonly, the block zone assumed to be present in these cases changes its spatial localization from time to time, with a simultaneous change of the areas responding to each centre; and that atrial dissociation while rare in the classical variety (type Schrumpf) is probably, in complex forms, widely present in human beings.

Summary

Two more cases with atrial dissociation (type Schrumpf) are reported. Previous clinical examples and experimental evidence of atrial dissociation are briefly discussed. Reasons are given for the assumptions that this phenomenon cannot always be considered as being due to an interatrial dissociation, that commonly the block zone between the independent atrial areas must be wandering, and that the complex forms of this pararrhythmia probably are much more common than has been widely accepted until recently.

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